1185-117 **Extended Distribution of Prolonged and Fractionated Right Atrial Electrograms Predicts the Development of Chronic Atrial Fibrillation in Patients With Idiopathic Paroxysmal Atrial Fibrillation**

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Background: The prolonged and fractionated atrial electrograms defined as a duration of 100 ms or longer and/or eight or more fragmented deflections are known to be the electrophysiologic characteristics in paroxysmal atrial fibrillation (PAF). However, the contribution of these abnormal atrial electrograms (AAE) to transition from PAF to chronic atrial fibrillation (CAF) is unknown.
Methods: Ninety-six patients (64 male, aged 57) with idiopathic PAF underwent electrophysiologic study (EPS) were followed. In EPS, 12 sites right atrial (RA) endocardial mapping (four aspects of the high, middle, and low RA) for detection of AAEs and RA extrastimulation for atrial vulnerability; repetitive firing, fragmented activity and conduction delay, were performed.
Results: During the follow up periods of 60-130 months, the development from PAF to CAF was observed in 17 patients (CAF group). Remaining 79 patients maintained in sinus rhythm with PAF (PAF group). The prevalence of AAE at middle RA was significantly high in CAF group, although high prevalence of AAE was observed at high RA in both groups (table). Kaplan-Meier analysis clearly showed that over 50% of the patients with AAEs at middle RA developed to CAF, on the contrary, only 7% were developed without those ($p<0.0001$). Indices of atrial vulnerability did not influence the transition to CAF.
Conclusion: These data suggested that the extended distribution of AAEs from high to middle RA was considered to be a predictive factor for the development from PAF to CAF.

Prevalence of Abnormal Atrial Electrograms

	high(%)	middle(%)	low(%)
CAF group	88.2	70.6	5.9
PAF group	63.3	13.9	6.3
p value	0.11	< 0.005	0.95

1185-118 **Pulmonary Vein Diameter is Increased in Patients With Paroxysmal Atrial Fibrillation Compared to Age-Matched Controls**

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Background: The pulmonary veins (PV) are important triggering sites in patients with idiopathic paroxysmal atrial fibrillation (PAF). The explanation for individual patients developing PV-triggered atrial fibrillation is unknown, but it has been suggested that PV dilatation and stretch may play a role. The purpose of this study was to compare the sizes of the proximal pulmonary veins in patients with PAF with those of patients without PAF.
Methods: 12 patients with PAF (age 52± 20 years, ejection fraction 57± 4%) underwent CT angiogram (CTA) of the chest (Picker 5000, intravenous injection of 140 cc of Ultravist 300, 3 mm collimation) prior to PV isolation procedure. PV diameter was measured at the junction of the PV with the left atrium. Similar measurements were performed in an age-matched control group of 10 consecutive patients with no history of AF, normal cardiac size and function, who underwent CTA of the chest to exclude pulmonary emboli.
Results:

	PAF (n=12)	Control (n=10)	P value
Left upper PV (mm)	14.7±2.3	12.0±1.3	<0.05
Left lower PV (mm)	15.3±3.0	11.8±1.3	<0.01
Right upper PV (mm)	15.3±3.2	12.0±1.7	<0.01
Right lower PV (mm)	16.4±3.0	12.4±1.0	<0.01

Conclusion: PV diameter, consistent in each of 4 PVs, in patients with idiopathic PAF is increased compared with age-matched controls. These observations support the concept that PV arrhythmogenicity might be related to stretch of PV myofibers.

1185-119 **Is Mode Switching a Surrogate Marker for Paroxysmal Atrial Tachyarrhythmias?**

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Background: In patients with tachycardia-bradycardia syndrome (TBS) and permanent pacemakers (PPM), mode-switching (MS) episodes are considered surrogate markers for atrial tachyarrhythmias (ATA). The purpose of this study is to evaluate the accuracy of the MS algorithm of Medtronic PPM for the detection of ATA. **Methods:** Patients with ATA implanted with dual chambered Medtronic Thera DR or Kappa series PPM for TBS were enrolled. MS was activated in all patients and programmed at nominal settings. Atrial sensitivity was ≥ 2.0 mV at implant and programmed to ≥ 4x sensing threshold. Serial Holter monitoring (HM) was performed and the fully disclosed tracings reviewed for time of onset, duration, and type of ATA. Pacemaker interrogation was performed at the end of each HM period. Date, time, and duration of each PPM MS episode were compared to those variables of the ATA as detected on HM. **Results:** Nineteen patients with a history of ATA and PPM for TBS received HM for a combined total duration of 1638.8 hours. Six patients had a total of 43 ATA episodes (38 atrial fibrillation, 5 atrial flutter) as documented on HM. PPM interrogation demonstrated appropriate MS in 42/43 (97.7%) ATA episodes. Sensitivity of the MS algorithm for duration of atrial fibrillation and atrial flutter was 100% and 95.5% respectively. Overall sensitivity for the duration of all ATA was 97.6%. Specificity for all ATA was 100%. Twenty-six inappropriate reversions of MS occurred; all instances occurred during atrial flutter and were the result of undersensing of flutter waves. **Conclusion:** In patients with TBS and PPM, the MS algorithm on the Medtronic Thera DR and Kappa 700 series is highly sensitive and specific for ATA. MS events are surrogate markers for ATA.

1185-120 **High Infusion Rate of Isoproterenol in Patients With Atrial Fibrillation: Frequent Evidence of Firing From Multiple Veins**

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We report the impact of increasing dose of isoproterenol infusion (ISO) on the ability to trigger atrial fibrillation (AF) in the Electrophysiology laboratory.
Methods and Results: One hundred eighty patients presented for mapping and ablation of AF using the circular mapping technique. Isoproterenol infusion was initiated at a dose of 2∝g/min for 5 minutes and then increased by 3 ∝g/min every 5 minutes and up to 20 ∝g/min in all patients after isolation of the culprit vein. After isolation of the culprit arrhythmogenic PV, firing from a different vein was seen with a mean ISO of 8.2±3.2 ∝g/min (range 6-10 ∝g/min) and 16.2±4.2 ∝g/min (range 10-20 ∝g/min) in 30 % (54/180) and 43% (77/180) of patients, respectively. These arrhythmogenic APCs originated from a different PV in 117 patients. A mean ISO infusion rate of 13±3 ∝g/min, 16±4 ∝g/min, and 18±3 ∝g/min could induce arrhythmogenic APCs from 2 PVs in 48 patients, from 3 PVs 29 patients, and from 4 PVs in 12 patients.
Conclusion: High infusion rates of isoproterenol appeared to increase the ability to trigger PVs firing. Evidence of firing from multiple veins during high dose ISO support the need to isolate all four PVs in order to maximize cure.

POSTER SESSION

1186 Predictors of Ventricular Arrhythmias

Tuesday, March 19, 2002, Noon-2:00 p.m.
Georgia World Congress Center, Hall G
Presentation Hour: 1:00 p.m.-2:00 p.m.

1186-107 **Exercise-Induced Microvolt T-wave Alternans in the Congenital Long QT Syndrome**

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Background: Macrovolt T-wave alternans (TWA) with alternating changes in the T-wave have been described in congenital Long QT Syndrome (LQTS). However, the presence of microvolt TWA has not been evaluated systematically in LQTS pts. **Methods:** We enrolled 67 consecutive LQTS pts (age = 38 ± 16 yrs; 43 F, 64%) from 30 distinct families and 78 unaffected control pts (age = 35 ± 11 yrs; 38 F, 49%) in a prospective exercise study. 20 LQTS pts were taking beta-blocking agents (BB) at time of study, and 3 had sympathectomy. No controls were taking BB or had coronary disease. TWA was measured at rest and during bicycle exercise testing (n=67 LQTS) or with atrial pacing (n=18 LQTS pts) by the spectral method using a CH2000 system (Cambridge Heart, Bedford, MA). If there was sustained alternans voltage > 1.9 ∝V and alternans ratio > 3 with an onset heart rate (HR) <120 beats/min (BPM) during exercise in either 1 orthogonal or 2 adjacent precordial leads for at least one minute which then persisted above that HR, the test was considered positive ("classic pattern TWA,' CP TWA). Max negative HR was defined as the highest interval HR at which sustained alternans was not present. Onset HR was defined as the HR above which CP TWA (> 1 minutes) was consistently present. **Results:** LQTS pts exercised 7.8 ± 2.4 min and achieved a peak HR of 149 ± 171 bpm. Control pts exercised 9.6 ± 3.3 min and achieved peak HR = 141 ± 21 bpm. Study pts reached a peak HR > 105 bpm for at least 1 minute with exercise or atrial pacing. Five LQTS pts (5/67, 7%) had classic pattern TWA with onset HR of 106 ± 5 bpm. One LQTS pts had CP TWA before and after BB. No control pts (0/75, 0%) demonstrated CP TWA ($p<0.05$). We defined transient pattern TWA (TP TWA) as presence of > 1 min microvolt TWA for exercise HR between 105-120 bpm which did not persist throughout exercise above the onset HR. Nine LQTS pts and 6 control pts had TP TWA. The max negative HR for LQTS pts was 112 ± 8 bpm and for control pts 121 ± 7 bpm ($p > 0.05$). 14/67 LQTS (21%) vs. 6/78 normals (8%) had either CP TWA or TP TWA ($p < 0.02$). **Conclusions:** (1) The presence of either transient or classic pattern microvolt TWA may be a new diagnostic tool for LQTS. (2) The classic pattern of microvolt TWA with bicycle stress testing alone is uncommon in congenital LQTS.